

1 A Oh, other people did studies and said, well, we
2 did not find that, so it must be wrong.

3 Q So there was a debate in the scientific
4 literature about whether Falck was correct?

5 A There was some disagreement about it, yes.
6 Now, I think now things are beginning to come back with
7 the congener specific analysis. They are going to come
8 back and probably validate what they put forward in '92.

9 Q What Falck, et al., put forward in '92?

10 A Yes.

11 Q But Falck and his co-authors were looking at
12 PCBs generally, not specific congeners?

13 A That's right. They looked at, as I said, a
14 summation technique.

15 Q And that was the technique that we saw cited in
16 later literature where the author said it didn't show
17 any association?

18 A Yes.

19 Q And what Falck actually does say is on page
20 145, "The finding of higher tissue levels
21 among cancer cases may also
22 signify a redistribution of chemicals to
23 the breast during the disease process."
24 Do you see that?

25 A Yes.

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1 risk; is that correct?

2 A No, they didn't. All they did is 20 patients
3 and 20 controls. It is not a population study. You
4 can't do relative risk. What you are doing here is a
5 biomarker study.

6 Q Simply showing a correlation between a level
7 of --

8 A Right. As they say in the brief introduction,
9 this class of compounds is a good candidate for being a
10 risk factor for breast cancer. That is why they looked
11 at it.

12 Q That is really all they are doing, to try to
13 find out if it is a risk factor?

14 A Right.

15 Q Not to find out to what extent its risk factor
16 or what dose level --

17 A No, there is no quantification intended or
18 implied here.

19 Q And --

20 A And what it does simply say is that the higher
21 the exposure, presumably the higher the risk.

22 Q Does it say that?

23 A No, I said it implies. That is the implication
24 of the study and that is why it created such a stir,
25 because it raised the possibility, oh, my God, there may

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1 Q Do you agree with that statement?

2 A I think subsequent events would indicate that
3 is probably not occurring.

4 Q That subsequent examination of specific levels
5 of specific congeners --

6 A Yes, where they looked at blood fat PCBs and --
7 you know, this was a tissue issue. There is no reason
8 to think that patients with breast cancer would have
9 higher blood PCB levels.

10 I mean, there is just no precedent for that.
11 In fact, there is not even any data to support the
12 notion that there is distribution of greater number of
13 PCBs into the breast tissue of a patient with breast
14 cancer. I mean, there is no biological support for that
15 notion.

16 Q But why do scientists look at breast tissue and
17 calculate PCB levels in breast cancer patients as
18 opposed to looking at -- I don't know -- legs or toes?

19 A Because breast tissue is a fatty tissue and the
20 chemical accumulates in fat.

21 Q Is lipid filled?

22 A Yes. But you are also interested in disease in
23 that organ. You want to know is there a concentration
24 in that organ of this chemical.

25 Q So Falck, et al, did not calculate relative

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1 be this chemical that is in every single person in the
2 United States.

3 It is in half the foods that we eat and it
4 causes breast cancer, maybe that is why the rate of
5 breast cancer has doubled in last 20 years.

6 Q PCBs are in half the food we eat?

7 A Oh, yeah, just like dioxins. Particularly in
8 farmed salmon.

9 Q Right. We talked about that.

10 A But there is a lot of other foods where it is
11 present not in such high amounts like in salmon, but it
12 is present.

13 THE WITNESS: Time for a break?

14 MR. HOPP: Let's take one.
(Brief recess.)
(Defendants' Exhibits 137 was marked for
17 identification by the court reporter.)

18 BY MR. HOPP:

19 Q Handing you what we have marked as Exhibit 137.
20 This is the Hansen article referenced in your report at
21 number seven under breast cancer; correct?

22 A Yes.

23 Q This actually talks about male breast cancer
24 after occupational exposure to gasoline and vehicular
25 combustion products; right?

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1 A Yes.
 2 Q What relevance does it have to Sherrie Barnes?
 3 A Well, that's a good question. The mechanism of
 4 breast cancer in men is possibly different than the
 5 breast cancer in women.
 6 I mean, men and women's breast cancer may have
 7 a different etiology. I think probably the important
 8 issue here is this would be support of these chemicals
 9 that were, in this case, particularly the benzene and
 10 the PAHs are present in our case here.
 11 And the implication of the study was that there
 12 was an increased risk that they thought was attributable
 13 to these exposures and this is a one case report.
 14 It is not terribly important to our overall
 15 case, but it is -- let's go to the last paragraph where
 16 he discusses this issue.
 17 He basically talks about, "The
 18 Elevated risk of breast cancer
 19 Among men, occupational exposed
 20 Gasoline and combustion products
 21 Has not been reported previously
 22 Except in one small study with
 23 nonsignificant odds ratio of 1.3.
 24 However, two recent studies show an
 25 increase in breast cancer in women

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1 to these chemicals that developed a rare disease.
 2 Oftentimes rare diseases, like men's breast
 3 cancer or mesothelioma can give us a lot of clues and
 4 should be followed up when they occur.
 5 Q Would it be fair to characterize the Hansen
 6 paper as generally informative, but not directly related
 7 to the cause of Sherrie Barnes' breast cancer?
 8 A Yes.
 9 Q In fact, the article does not calculate the
 10 relative risk for breast cancer in women; is that right?
 11 A Correct.
 12 Q And at what exposure level does the study
 13 indicate that breast cancer has increased in men?
 14 A Well, he has got an odds ratio here of 2.2 with
 15 no lag time and 2.5 with ten years of lag time with
 16 statistical significance.
 17 Q Lag time being years of exposure? What does
 18 lag time mean?
 19 A No. What that does is it allows for more
 20 latency.
 21 Q Okay.
 22 A In other words, you look at the people's
 23 exposure and then you make sure that you are at least
 24 allowing for ten years of lag time from the time of
 25 exposure to the time of the disease diagnosis.

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1 exposed to benzene and PAHs."
 2 Which is the Petralia study, I believe is also
 3 on this list. I know it is on my new list.
 4 And it is here on this list, and then he
 5 states, "The similarities among some of
 6 The known risk factors for breast
 7 Cancer in men and women and a
 8 Similar variation in incidents
 9 Point to common etiologic factors;
 10 therefore, gasoline and combustion
 11 products caused breast cancer in
 12 Men. It probably does so in women,
 13 too."
 14 And then it goes on to discuss some other
 15 things.
 16 Q So the author is hypothesizing that this result
 17 that he obtained in this paper might be applicable to
 18 women, as well; is that fair?
 19 A Yes, and then he alludes to some other studies
 20 that showed he doesn't do an exhaustive review. Where
 21 we actually know that there are other papers that he
 22 could have cited.
 23 Q Sure. And we will get to those.
 24 A But the point is that it is just another study
 25 of a case of someone who has some pretty good exposures

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1 Q Is it accurate to say that the Hansen study
 2 doesn't examine specific exposure levels, but rather
 3 looks at occupational exposure of gasoline and
 4 combustible products in general?
 5 A Yeah, 230 male employees were members of the
 6 National Pension Fund and the country is Denmark. And
 7 he looks at job title for exposure.
 8 Q Okay. So there is no exposure data for the
 9 individual study subject?
 10 A No.
 11 Q Next one on your list -- your breast cancer
 12 reference list number eight is the Holford,
 13 H-O-L-F-O-R-D, study?
 14 A Yes.
 15 Q Handing you what we have marked as Deposition
 16 Exhibit No. 138. This is a copy of the Holford study.
 17 The Holford study is entitled Joint Effects of Nine
 18 Polychlorinated Biphenyl (PCB) Congeners on Breast
 19 Cancer Risk; is that right?
 20 (Defendants' Exhibits 138 was marked for
 21 identification by the court reporter.)
 22 THE WITNESS: Yes.
 23 BY MR. HOPP:
 24 Q And Holford looked at nine PCB congeners;
 25 right?

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<p>1 A Yes.</p> <p>2 Q And, generally, what did Holford conclude?</p> <p>3 A There is an association with some of them.</p> <p>4 Let's see if I can make sense out of this.</p> <p>5 Table 2 shows odds relative risk associated</p> <p>6 with a ten PB change in exposure to individual congeners</p> <p>7 by type of model; and I think the risk associated</p> <p>8 congener values that are listed in the middle .2153 and</p> <p>9 156 is not being significant.</p> <p>10 Q Okay.</p> <p>11 A But 183 is significant.</p> <p>12 Q 180 is slightly elevated, but not significant;</p> <p>13 right?</p> <p>14 A Yes, 180 is slightly elevated, but it is not</p> <p>15 very significant. It is almost significant, it is .99.</p> <p>16 It is real close. Anyway --</p> <p>17 Q But 183 is the culprit in that Holford paper;</p> <p>18 right?</p> <p>19 A That is the one that they felt was</p> <p>20 statistically significantly associated.</p> <p>21 Now, on Table 3, they give an odds ratio</p> <p>22 associated with a level of PCB in quintiles and they</p> <p>23 divided them into five levels.</p> <p>24 Q I'm sorry. Table 3?</p> <p>25 A Table 3, it is at the bottom of 979.</p> <p style="text-align: right;">800</p>	<p>1 included in linear logistic model."</p> <p>2 Q What the heck does that mean? Do you</p> <p>3 understand that?</p> <p>4 A Yeah, they are doing statistical analysis,</p> <p>5 which is -- when you have multiple variables like this,</p> <p>6 you know, a dozen or so PCBs, plus other variables, age</p> <p>7 and whatever else you put in the model, you have got a</p> <p>8 very complex statistics; but not being a statistician, I</p> <p>9 cannot really explain to you what they are doing. It is</p> <p>10 a very high order statistical.</p> <p>11 Q Well, principal component analysis is the</p> <p>12 general name for what they did?</p> <p>13 A Yes. Well, that component.</p> <p>14 Q All right.</p> <p>15 A In the Statistical Methods, they discuss their</p> <p>16 analysis, how they did it, and one they want to look at</p> <p>17 the joint effects of individual PCB congeners on the</p> <p>18 risk of breast cancer and whether the effect of each</p> <p>19 congener was the same, which was tested using linear</p> <p>20 contrast.</p> <p>21 "If these results suggested</p> <p>22 That the magnitude of effect on</p> <p>23 Breast cancer risk was different</p> <p>24 From the congeners, then it</p> <p>25 Would not make sense to evaluate</p> <p style="text-align: right;">802</p>
<p>1 Q Okay.</p> <p>2 A And there, they -- as the level of the PCB</p> <p>3 increased, the odds ratio of relative risk -- I think it</p> <p>4 is related risk score, which is similar to relative</p> <p>5 risk, it is adjusted estimates of relative risk. Risk</p> <p>6 becomes statistically significant only at the top</p> <p>7 quintile. Otherwise, the curve is pretty flat.</p> <p>8 Q And Table 3 is looking at all of the congeners</p> <p>9 that are being studied or is it --</p> <p>10 A They have some kind of PCB score. It is a</p> <p>11 score -- let's see how they scored it. Somewhere in</p> <p>12 here they describe the score.</p> <p>13 All right. Well, it is on Page 977. It is</p> <p>14 called Principal Components, and they describe what they</p> <p>15 did.</p> <p>16 "In order to understand better</p> <p>17 the nature of the effects for</p> <p>18 individual congeners, principal</p> <p>19 components analysis was used</p> <p>20 to create factors that were</p> <p>21 independent of each other.</p> <p>22 Using PRO PRINCOMP in SAS we</p> <p>23 estimated the eigenvectors, which</p> <p>24 provided loading scores that gave</p> <p>25 rise to new variables to be</p> <p style="text-align: right;">801</p>	<p>1 Total PCB exposure, but to</p> <p>2 Investigate the joint effects of each</p> <p>3 congener. Regression diagnostics</p> <p>4 Were used to determine whether:</p> <p>5 The results were sensitive to one or</p> <p>6 more influential observations."</p> <p>7 Q I'm sorry.</p> <p>8 A Now, we are talking about regression</p> <p>9 diagnostics was used on one or more influential</p> <p>10 observation.</p> <p>11 "But the overall conclusions</p> <p>12 Were found to be stable. Bootstrap</p> <p>13 methods were used to estimate</p> <p>14 Bias in the estimates of risk, as well</p> <p>15 as providing alternative estimates of</p> <p>16 standard errors. While the resulting</p> <p>17 standard errors were slightly greater,</p> <p>18 the conclusions were essentially</p> <p>19 unchanged, so these results are not</p> <p>20 present."</p> <p>21 I think what they are saying is that their</p> <p>22 principal component analysis is what they used and that</p> <p>23 is what they used in Table 3 as a related risk score.</p> <p>24 Q And above Table 2, the authors point out, the</p> <p>25 statement is, "Notice that some congeners</p> <p style="text-align: right;">803</p>

1 Are positively associated with breast
2 cancer risk, while others are negative";
3 is that right?

4 A Well, if you look at the standard coefficient,
5 the first line, when it says, negative .021, that means
6 that the higher the PCB level of the congener, the lower
7 the breast cancer risk.

8 Q All right.

9 A So that is right. There were three – four
10 that were negative and then one, two, three, four, five
11 that were positive.

12 And 180 was the most positive statistical and
13 it reached almost statistical significance and 183 did.

14 Q And in the Discussion section, this is on Page
15 979, the authors point out that, "The
16 Association of total PCB exposure with
17 breast cancer risk in this analysis was
18 estimated to be small and inverted."
19 Is that what you are talking about?

20 A Yes, for those who had it – the higher the
21 level, the lower the risk, suggesting – I think, you
22 know, you can find and do these fancy statistics. You
23 can find things like this. That may not mean anything.
24 The most important thing here is to look at all
25 the congener correlations, and 180 and 183, again,

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1 correlates strongly with the total congeners.
2 In other words, you are getting a positive
3 effect on the breast cancer. And like the other studies
4 we have looked at, if you add up all of the PCB
5 congeners, that also correlates with breast cancer risk.
6 So what it would suggest is that the overall
7 mixture, maybe some components being more important than
8 others, is contributing to the risk; and that the
9 negative components do not outweigh the positive
10 components in terms of causing the effect that we are
11 seeing in the increased risk.

12 Q But they do balance out and that is why the
13 authors say that the overall risk is small?

14 A That's correct.

15 Q And they go on in the Discussion section and
16 say, "These results suggest that some
17 Congeners have a protective effect on
18 breast cancer risk, while others are
19 associated with an increased risk"; is
20 that right?

21 A That's right. That is correct.

22 And I think that is consistent with all the
23 data. It shows that there is a small but significant
24 increase in risk. And the reason it is important is
25 that there are so many darn people exposed and so many

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1 people get this disease, that anything that contributes
2 to the risk is important to address.

3 Q Is this a case control study?

4 A This is a biomarker study. I mean, there is
5 cases and controls. What they are doing is they are
6 studying the presence of a biomarker in PCBs in two
7 populations to see if the testing hypothesis that the
8 cases would have a higher level of these chemicals than
9 the controls.

10 And the answer is, yes, and it does show
11 correlation.

12 Q Does this study indicate what dose of any
13 particular PCB congener is necessary to cause an
14 increased risk of breast cancer?

15 A No, I mean, if you look at the – I don't think
16 there is a single measurement in this whole paper. It
17 is all statistical analysis.

18 Let me just see. Maybe they are mentioned
19 somewhere. The level – no, what they are really trying
20 to do is the correlation or the association of the
21 chemical versus the risk. And that is not going to give
22 you thresholds or slope factors.

23 Q The next paper in order on your reference list,
24 this is number nine, is the Hoyer paper; is that right?

25 A Yes.

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1 Q I am handing you what we have marked as
2 deposition Exhibit No. 139.
3 (Defendants' Exhibits 139 was marked for
4 identification by the court reporter.)
5 BY MR. HOPP:

6 Q Is this the Hoyer paper?

7 A Yes.

8 Q And it is entitled Organochlorine Exposure and
9 Risk of Breast Cancer. What question was Hoyer trying
10 to answer?

11 A The same question. He looked at Dieldren,
12 which is an organochlorine. He looked at
13 chlorocyclohexane, which is another pesticide,
14 organochlorine pesticide.

15 Q Did this study look particularly at TCDD or
16 dioxin?

17 A No, it looked at PCBs, DDE, but it did not look
18 at dioxin per se.

19 Q So this would be another study that is
20 generally informative, but it is not directly related to
21 Sherrie Barnes; is that right?

22 A Yes. For the reasons that I indicated earlier,
23 I thought it was relevant.

24 Q And they actually looked at serum levels; is
25 that correct?

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1 A Yes.
2 Q So these are blood samples and not tissue
3 samples?
4 A Yes, serum sampling. That is right.
5 Q The Result section indicates that,
6 "The risk of breast cancer decreased
7 with increasing number of full-term
8 pregnancies and increased with" -- I'm
9 sorry -- "and increasing with body
10 weight and height."
11 Do you see that?
12 A Where are you reading from?
13 Q The Result section; this is Page 1818 starting
14 right above that table.
15 A "Increasing number of full-term
16 Pregnancies and increasing with
17 Body weight and height."
18 So height was made a standard.
19 Q You wouldn't think so. But Hoyer at least
20 concludes that increasing body weight and height are a
21 risk factor; is that right?
22 A This is the first time I have ever seen height
23 as a risk factor for anything. And unmarried women had
24 an 89 percent higher risk than married women. It is
25 probably because they didn't have babies.

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1 Q Moving on down this page, this is 1818. It
2 says, "We found a slight increase in
3 Risk of breast cancer with increasing
4 concentrations of BHCH, but no
5 association was apparent for total
6 DDT or total PCBs."
7 Do you see that?
8 A Um-hmm.
9 Q So this study tends to conflict with some of
10 the other studies which have indicated PCBs increase the
11 risk of breast cancer?
12 A Well, they did 28 PCBs. They don't tell us
13 which ones. So this wasn't as detailed a congener
14 analysis as the others.
15 They do list them here. And -- yes, they just
16 didn't find a correlation.
17 Q And then the Conclusion, which is on the last
18 page states, "Our results support the
19 Hypothesis that organochlorine
20 compounds, such as dieldrin,
21 Which have oestrogenic properties,
22 May increase the risk of breast cancer.
23 They do not, however, suggest that
24 exposure to total PCB, total DDT,"
25 And I guess, "P prime-DDE have any

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1 influence on the risk of breast cancer."
2 Is that right?
3 A Yes, in this study, they did not find an
4 increase in breast cancer. That's correct.
5 Q The next study in order under List of Breast
6 Cancer References is the -- maybe you can pronounce it
7 for me. Kogevinas paper?
8 A Kogevinas is as good as any.
9 Q Kogevinas, K-O-G-E-V-I-N-A-S.
10 I am handing you what we have marked as
11 deposition Exhibit No. 140, which is the Kogevinas
12 paper.
13 (Defendants' Exhibits 140 was marked for
14 identification by the court reporter.)
15 BY MR. HOPP:
16 Q Now, this is a review article; is that right?
17 A It is.
18 Q So it doesn't report on a new experiment, but
19 rather discusses studies done by other people?
20 A Yes.
21 Q And does Kogevinas find -- well, let me -- what
22 does Kogevinas conclude, generally, based on the other
23 studies?
24 A More studies are needed. That was his main
25 conclusion, but he reviews some of the studies and it is

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1 interesting in that respect.
2 Q And that is, again, generally informative, but
3 not particularly relevant to Sherrie Barnes?
4 A Correct. He gives a list of the various
5 studies and notes, you know, the breast cancer,
6 including male breast cancer, has been found to be
7 increased.
8 Q He finds increasing mortality from breast
9 cancer that is not statistically significant; is that
10 right?
11 A Yes.
12 Q That is in Table 5?
13 A Yes. Table 5 he is looking at -- where is
14 that? He has got different references 170 -- where is
15 it? I am trying to see what his references are for
16 that.
17 Anyway, he -- I guess, IARC's international
18 cohort study of phenoxy herbicides or chlorophenols
19 where TCDD was presumed to be present and the SMRs are
20 elevated for all of the cancers, but all malignant
21 neoplasms are statistically significantly increased.
22 And the individual types of cancer, breast
23 female is almost statistically significant. The odds of
24 SMR is 2.16, but the confidence interval is at .99. We
25 are talking about 100ths off. Otherwise, it would be

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1 statistically significant.
2 So that, in light of all the other evidence we
3 have, this is supportive.
4 Q All right. But you are looking in this paper,
5 Table 5, you are looking at nine deaths; is that right?
6 A Yes.
7 Q Out of how many expected?
8 A Well, that would be 2.16 more than expected.
9 So you would expect in that population -- I guess, the
10 174 reflects the number of something rather -- what is
11 it? I don't know the number of people at risk; but they
12 expected half of that many cases. So there is a
13 doubling of risk.
14 Q If the spread at the 95 percent confidence
15 interval includes one, then it is not statistically
16 significant?
17 A Yeah, I know. And if it was one more, it would
18 be.
19 That is the point I am trying to make is it is
20 very close to statistically significant; but if the
21 numbers were bigger, it would be significantly.
22 And as I say, by itself, it would not be
23 important, but taken in light of all of the other
24 evidence, it is supportive.
25 Now, the same is true of male breast cancer.

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1 It has doubled 2 1/2 times the -- twice as they
2 expected.
3 And, again, that goes along with our other
4 observations about this and, similarly, prostate is
5 elevated. Testes is elevated. Thyroid is elevated, and
6 all endocrine organs are elevated. The numbers are
7 small.
8 Q And not statistically significant?
9 A Not statistically significant, but the point is
10 all of these cancers are endocrine disruption sensitive
11 cancers. And, again, in view of other information, it
12 certainly is worth paying attention to.
13 Now, if you go over to the last one,
14 "All workers exposed to any phenoxy
15 herbicide or chlorophenyl."
16 Q Still on Table 5; right?
17 A Still on Table 5. You have got a statistically
18 significant excess of, again, all malignant neoplasms
19 and other endocrine organ cancers are elevated
20 statistically significant.
21 So it would appear to me that, you know, this
22 paper is useful.
23 Q In a general way?
24 A Correct.
25 Q It does not identify a particular dose level

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1 which is required to increase the risk of breast cancer;
2 is that correct?
3 A No.
4 Q The next paper in order on your list of breast
5 cancer references is the Laden paper, L-A-D-E-N; is that
6 right?
7 A Yes.
8 Q And it is number 11; correct?
9 A Yes.
10 Q I am handing you what I have marked as
11 Deposition Exhibit No. 141.
12 (Defendants' Exhibits 141 was marked for
13 identification by the court reporter.)
14 BY MR. HOPP:
15 Q This is the Laden paper; is that right?
16 A Yes.
17 Q And the Laden paper looks at the Nurses' Health
18 Study; is that right?
19 A Yes.
20 Q Is that otherwise sometimes called the Harvard
21 Nurses' Study?
22 A Well, this is from Harvard. So it could be
23 considered the Harvard Nurses' Study.
24 Q Have you heard that expression before, the
25 Harvard Nurses' Study?

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1 A No. I heard the Harvard Doctors' Study, but
2 yesterday when you mentioned the Harvard Nurses, this is
3 the first I heard of it.
4 But as I said, this is a study of nurses
5 conducted by Harvard. So it would be appropriately
6 called that.
7 Q And correct me if I am wrong, but it appears
8 that what happened was Harvard or some group at Harvard
9 has collected and has continued to collect data on a
10 large group of nurses.
11 It is sort of a prospective study. It examines
12 health effects over the course of the lives of these
13 women?
14 A Yes, just like the doctors' study. Same idea.
15 Q The idea is to --
16 A Follow the large group and see what happens to
17 them and look at the different risk factors
18 prospectively.
19 Q It states, at the end of the abstract,
20 "The majority of studies have concluded
21 the exposure to PCB are unlikely to be a
22 major risk factor for breast cancer."
23 Is that right?
24 A Are you talking about --
25 Q I am looking at the end of the abstract.

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1 A Although there is no independent association,
2 blah, blah, blah -- yeah, the point of this paper is
3 that if you look at the nurses who have this particular
4 polymorphism, CYP1A1-exon 7, this is a risk factor for
5 breast cancer.
6 Q Okay.
7 A And I think what they found was --
8 Q Was what he found that this was a genetically
9 susceptible population?
10 A Correct.
11 Q Okay. Doctor, do you want to continue with
12 your answer?
13 A What they say here is, "However
14 High levels of PCBs may be associated
15 with breast cancer risk in the subgroup
16 of women who have variant
17 CYP1A1-exon 7 polymorphism."
18 Additional studies are needed to examine
19 that possibility.
20 Q That is CYP1A1-exon 7 polymorphism, that is
21 something to do with the particular genetic structure of
22 these women; is that right?
23 A Yes.
24 Q It is a gene?
25 A Their ability to transform the PCBs or handle

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1 or not it would be relevant to test her.
2 Q This study looks at latent PCBs; correct?
3 A Correct.
4 Q Doctor, I have handed you what we have marked
5 as Exhibit 142. This is the next reference on your
6 breast cancer list. It is number 12 and the author is
7 Leis or Lees. L-E-I-S.
8 (Defendants' Exhibits 142 was marked for
9 identification by the court reporter.)
10 THE WITNESS: Yeah.
11 BY MR. HOPP:
12 Q And this is really just a paper on diagnosing
13 breast cancer; is that right?
14 A Yes, it has risk factors. That is the reason
15 it is here.
16 Q But does it talk about environmental risk
17 factors or TCDD?
18 A Not really, it talks -- Table 1 and Table 2,
19 exogenous estrogen, which would be in birth control
20 pills and hormone replacement. And then it says,
21 "Carcinogenic exposure,
22 particularly to viral agents
23 and some drugs."
24 Q So --
25 A Really, it just kind of gives you a list of

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1 them is impaired or reduced.
2 Q And that's the only study -- strike that.
3 That is the only population in which the Laden
4 paper found an effect with increased levels of PCB; is
5 that right?
6 A That's right.
7 Q And we don't know whether Sherrie Barnes had
8 that particular polymorphism, do we?
9 A No. You asked me that yesterday. So we don't
10 have any studies on Sherrie Barnes or anybody else on
11 cohort. It is not a routine thing you send to the lab.
12 Q You have to take a tissue sample?
13 A You have to do genetic studies. That is what
14 you have to do to find this particular variant. It is
15 expensive and it is possible to be done. But it is very
16 important particularly in people that we don't have
17 disease in yet; but we want to know who is at high risk.
18 These kind of studies would be highly relevant.
19 Q Would it be possible to test Kenesha Barnes to
20 find out whether her mother had that particular
21 polymorphism?
22 A Well, we would have to check her dad, too. I
23 don't know how the inheritance goes for that particular
24 gene. I don't know if it can be an acquired defect. I
25 would have to study it to answer that question whether

817

1 things that have been raised as -- just kind of a
2 general review of the disease. So you know what you are
3 talking about.
4 Q Not very informative with respect to causation?
5 A Correct. I don't think he has references for a
6 lot of those causative factors. He doesn't give a
7 reference. He makes the assertion in this table.
8 Q The next breast cancer reference that you have
9 in order, number 13, Lucena, L-U-C-E-N-A; is that right?
10 A Right.
11 Q I am handing you what we have marked as
12 Exhibit 143.
13 (Defendants' Exhibits 143 was marked for
14 identification by the court reporter.)
15 BY MR. HOPP:
16 Q This is the Lucena paper; is that right?
17 A Yes.
18 Q It is entitled Short Communication. Is this --
19 is there some significance to that?
20 A Well, what they do is they write a very brief
21 paper presenting one table, maybe, which they think is
22 important when they want to publish it as a -- quickly,
23 so it is easier for the reviewers to deal with a short
24 paper with very little information, so you can get it
25 published faster.

819

1 Q Right. And this paper really identifies one
2 specific congener or PCB?
3 A 28.
4 Q As associated with an increase risk of cancer;
5 is that right?
6 A Yes. Fascinating. It is like every paper has
7 a different congener. Congener of the week.
8 Q Did Lucena look at other congeners?
9 A They looked at a bunch of them. It is listed
10 on the top of 118, left-hand column.
11 Q But the only one they found that significantly
12 increased the risk of breast cancer was 28; correct?
13 A Yes.
14 Q Once again, they think there is a great need
15 for more studies?
16 A That's, as I told you, every study will say
17 that. It is the stock and trade of a researcher.
18 Q Does Lucena calculate a relative risk for
19 exposure to PCB 28?
20 A Yes, 9.597, huge odds ratio. Same thing.
21 Q But it does not identify a particular dose
22 level for that congener which would result in that
23 increase risk; is that correct?
24 A I don't see that it was quantified. What they
25 said was in the difference between the exposed and the

820

1 controls, it was a ninefold difference in that chemical.
2 Q So what they were — this was a study in Spain;
3 is that right?
4 A Yes.
5 Q And they were actually looking at breast tissue
6 that had been removed from women who had breast cancer;
7 correct?
8 A Yes, that's correct.
9 Q And these were malignant lesions?
10 A Well, in the exposed, they were malignant.
11 Q And the controls, they were benign lesions; is
12 that right?
13 A Benign lesions.
14 Q So what they found was that if someone had a
15 detectible level of PCB 28 in the malignant lesion,
16 those people turned out to have a 9.597 odds ratio; is
17 that correct?
18 A That's right.
19 Q How is this paper — strike that.
20 How does this paper relate to or inform your
21 opinion with respect to Sherrie Barnes?
22 A The same as the other PCB papers. We are
23 talking about a similar toxicity for dioxin-like
24 chemicals.
25 Q The next one in order on your breast cancer

821

1 reference list is the Manz paper; is that right,
2 M-A-N-Z.
3 A Yes.
4 Q I have marked that deposition as 144. This is
5 the Manz paper; is that correct?
6 (Defendants' Exhibits 144 was marked for
7 identification by the court reporter.)
8 THE WITNESS: Manz paper, correct.
9 BY MR. HOPP:
10 Q M-A-N-Z?
11 A M-A-N-Z, from Germany.
12 Q This is a German study of exposure, actually,
13 of workers in a chemical plant; is that right?
14 A That's correct.
15 Q And they characterized — first of all, it is a
16 retrospective mortality study; correct?
17 A Yes.
18 Q And they characterized the herbicide workers in
19 this plant in Germany as being having been exposed to
20 heavy contamination of 2, 3, 7, 8-TCDD?
21 A Yes.
22 Q But only seven percent of the women worked in
23 high exposure areas of the plant; is that right?
24 A Yes.
25 Q Did they detect an increased risk of breast

822

1 cancer as a resulted of heavy exposure of 2, 3, 7,
2 8-TCDD?
3 A I think this is overall, the SMR for carcinoma
4 of the breast was 2.15 with a 95 percent confidence
5 interval of 0.98.
6 Again, right at the borderline, and 409 for
7 nine deaths.
8 Q And this is what table?
9 A It is on Page 962 under Mortality Among Women.
10 Q All right.
11 A Malignant neoplasms were right at not
12 significant, but the breast cancer was. And that's
13 really the point of the paper, which is about TCDD.
14 Q Okay. So it is just about TCDD, and the 2.15
15 is an increased SMR, but is it statistically
16 significant?
17 A Well, it is right at that borderline at 0.98.
18 Q Again, the 95 percent confidence level includes
19 one?
20 A That's right. It is right at the borderline.
21 Again, I think I have said it before, when it
22 is taken into the context of everything else, it is
23 supportive. They also review a study, which I don't
24 think we got, but —
25 Q Which study is that?

823

<p>1 A I am just looking at it here. I am wrong. 2 That's the only point of this study. 3 Q Does the Manz paper identify a dose level of 2, 4 3, 7, 8-TCDD, which is significant for increasing the 5 risk of breast cancer? 6 A No. 7 Q Is the exposure level documented in the Manz 8 paper? 9 A No, they don't do blood levels or the chemical 10 plant was found to have high TCDD levels enough to cause 11 chloracne. And that was led to the change in practices 12 to reduce exposures. 13 Q So qualitatively, they think it was high 14 because of the chloracne? 15 A Well, we know that when you get chloracne, you 16 are at high levels; but they don't give the numbers in 17 here. 18 Q While we are on the subject of chloracne, I 19 know I discussed this with Dr. Sawyer, and forgive me if 20 I covered this with you. 21 Are you familiar with the case of Victor 22 Yushchenko? 23 A Yes, I am. 24 Q Victor Yushchenko is the president of the 25 Ukraine; is that right?</p> <p style="text-align: right;">824</p>	<p>1 poisoning; correct? 2 A Not yet. 3 Q How about the women in Vienna, Austria; did 4 they -- 5 A They have not died yet either, but they are 6 being followed. They are about ten years from the onset 7 of exposure. And -- 8 Q Did they -- 9 A They are quite ill and I suspect Victor 10 Yushchenko is quite ill. They have been attempting to 11 get the levels down using various techniques to 12 detoxify, but nothing is working. But the levels of 13 both the two women from Austria and Yushchenko are still 14 extremely high. 15 Q They were using Olestra, I think, with 16 Yushchenko; is that right? 17 A They used Olestra with the two ladies from 18 Vienna, also. 19 Q Did it work? 20 A It is a miserable, miserable drug. It causes 21 diarrhea and people can't take it. So they take it for 22 a while till they get sick of it. It may lower the 23 level a bit. It is not terribly effective. 24 Q Olestra is the fake fat; right? 25 A That's right. The non-absorbable fat. It is</p> <p style="text-align: right;">826</p>
<p>1 A Yes, he is. 2 Q He was actually -- someone tried to poison him 3 with dioxin? 4 A That's right. 5 Q Do we know if it was 2, 3, 7, 8-TCDD? 6 A No, we don't know precisely, but he had dioxin 7 poisoning. And in the poisoning episode, they usually 8 use TCDD because it is available. If you are running a 9 lab when you are testing this, you can get TCDD as a 10 standard. 11 Q You could have gone to the German factory and 12 seen the Manz paper and gotten it? 13 A Yeah, I guess so. You can get purified TCDD 14 from a chemical supply house. 15 Q Now, the acute exposure to -- strike that. 16 I believe Dr. Sawyer testified that the level 17 of Victor Yushchenko's exposure to TCDD was among the 18 highest ever recorded? 19 A Among the highest recorded, that is correct. 20 Q There were a couple of other acute poisoning 21 cases that were documented, several women 20 years ago 22 or so, who were up in that range, as well; is that 23 right? 24 A Yes, from Vienna, Austria. 25 Q But Victor Yushchenko did not die from his</p> <p style="text-align: right;">825</p>	<p>1 the same as Cholestyramine and the cold pressed oils 2 that we use. It compresses (phonetic) in the gut. 3 Q And the women from Vienna, have they developed 4 breast cancer? 5 A No, not yet. And we talked about this earlier, 6 it may not be TCDD in the adult that causes the breast 7 cancer, anyhow. Or it may not be nearly as potent a 8 factor in the equation. I mean, you can induce -- 9 Q Let's move on. The next paper you have cited 10 in your breast cancer references, it is number 15, the 11 Morris paper; is that correct? 12 A That's right. 13 Q I am handing you what I have marked as 14 Deposition Exhibit No. 145. This is the Morris paper; 15 correct? 16 (Defendants' Exhibits 145 was marked for 17 identification by the court reporter.) 18 THE WITNESS: Yes. 19 BY MR. HOPP: 20 Q And this paper is -- would it be accurate to 21 call the Morris paper a hypothesis-generating paper? 22 A Well, he reviews all the data. That's the 23 value of reading a paper like this. 24 Q What, if anything, does Morris conclude? 25 A Well, he talks about benzene, benzopyrene. He</p> <p style="text-align: right;">827</p>

1 does talk about cigarettes, aromatic hydrocarbons, and
2 breast cancer, and PAHs.
3 Q Is Morris a review paper?
4 A Yes. He goes on to talk about PAHs and in
5 quite a bit of detail. And then concludes, you know,
6 that something going on in our environment is causing
7 this. And his candidate is aromatic hydrocarbons, in a
8 broad sense.
9 And he reviews a bunch of them. And, of
10 course, PAH is at the top of the list here. He does not
11 go into much detail on the polychlorinated hydrocarbons.
12 He is mainly focused on the aromatic
13 hydrocarbons. It is a very thorough review of those
14 papers up to that time.
15 Q It is sort of a dated paper; right, this is
16 '92?
17 A '92, but there was still quite a bit more
18 evidence already at that time.
19 Q Morris identifies radiation and aromatic
20 hydrocarbons as inducing and promoting mammary cancer;
21 is that correct?
22 A Yes, that's correct.
23 Q He also states that such disparate factors as
24 urban residents, geographic location of residents, and
25 life-style factors, such as alcohol ingestion, high

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1 polyunsaturated fat diet, and food selection and
2 preparation all contribute to exposure to promoter and
3 initiating influence of aromatic hydrocarbon
4 carcinogenesis; is that right?
5 A That's correct. That is what he says.
6 Q Does Morris isolate any particular exposure,
7 any particular PAH which he thinks is significant for
8 causing breast cancer?
9 A Benzopyrene and DB(AH)A anthracene, which are
10 the experimental animal carcinogens. He also mentioned
11 DMBA and PAHs in general.
12 Q In his review, does he discuss human
13 epidemiology studies, or any the animal studies and in
14 vitro studies?
15 A Well, he does -- he touches on animal studies
16 quite a bit. Because in '92, there were fewer studies,
17 but he mentions benzene, as well, and its ability to
18 induce cancer, and talks about the -- mostly the animal
19 study.
20 There wasn't as many studies back at that time
21 in humans as there are now. But he gives a background
22 as to why people started looking so hard at human
23 studies, subsequently.
24 And he points out why these chemical PAHs, in
25 particular, are likely to be the cause of breast cancer.

829

1 Q He does not address creosote as a mixture;
2 correct?
3 A No, he doesn't.
4 Q And does he document any exposure levels to any
5 particular PAHs?
6 A No.
7 Q Does he calculate relative risk levels?
8 A No, he doesn't do that either. This is a
9 review paper of pointing out all of the papers that
10 exist at that time that point towards a link between the
11 PAHs and breast cancer.
12 Q I understand, but Morris does not identify any
13 particular exposure level that is necessary to produce
14 harm; correct?
15 A No.
16 Q I'm sorry. That was a bad question.
17 Does Morris identify a particular exposure
18 level that is necessary to produce harm?
19 A No, he doesn't.
20 Q The next paper on your list of breast cancer
21 references is number 16, Muscat, M-U-S-C-A-T; correct?
22 A Yes.
23 Q I am handing you what we have marked as
24 Deposition No. 146.
25 (Defendants' Exhibits 146 was marked for

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1 identification by the court reporter.)
2 BY MR. HOPP:
3 Q This is the Muscat paper; correct?
4 A Yes, it is.
5 Q Entitled Adipose Concentrations of
6 Organochlorine Compounds and Breast Cancer Recurrence in
7 Long Island, New York; right?
8 A Yes.
9 Q So, again, he is looking at PCBs; right?
10 A Yes.
11 Q And what, if anything, does Morris conclude?
12 A Muscat.
13 Q I'm sorry. Muscat conclude?
14 A That there is a linkage between adipose PCB
15 levels, which is -- let me see. I think it is
16 recurrence in -- of breast cancer.
17 Q Muscat is looking at cancer coming back a
18 second time?
19 A Yes, he is talking about it being a predictor
20 of recurrence of breast cancer. Interesting study.
21 Q How does this relate to Sherrie Barnes?
22 A Again, it is showing PCBs which are dioxin-like
23 in their behavior increasing the risk of recurrent
24 cancer, which is relevant to our patient, I believe, in
25 the sense that she had a tumor that was very aggressive.

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<p>1 What they are suggesting here is that PCBs 2 probably increased breast cancer risk, but that is not 3 the main point. The main point is that it is – or 4 associated with recurrence. 5 Q So Sherrie Barnes had it once and it was fatal? 6 A Yes. She did not have a recurrence. She did 7 not respond to the therapy either, suggesting that her 8 tumor was very aggressive and malignant. 9 And this paper suggests that making the tumor 10 grow more readily would be associated with these types 11 of exposures. 12 Q And in the concluding paragraph, they point out 13 that these results, that is, the results represented in 14 deposition Exhibit 146, conflict – I'm sorry – 15 contrast with the author's previous data showing no 16 effect of organochlorine compounds on breast cancer in 17 these women; is that right? 18 A Yes. 19 Q So there was a previous paper by the same 20 authors which was negative; correct? 21 A That's right. 22 Q Does the Muscat paper calculate relative risk 23 of recurrence? 24 A Let's see, relative risk is on Table 5, and as 25 the level grows, most of the relative risk grows</p> <p style="text-align: right;">832</p>	<p>1 patients all the way – there are three or four that 2 aren't. 3 And no one stands out, but then the total turns 4 out to be statistically significant. And the biggest 5 difference is in the blacks. Where they – it is 129 6 parts per billion difference. 7 Q Can you explain that? What does it say about 8 black women? 9 A That blacks with no recurrence, their PCB total 10 was 406. The blacks with recurrence, their PCB level 11 was 529. Both of those values were higher than the 12 whites. 13 And the highest at all are the Asian with no 14 recurrence, but there is very small number of Asian, 15 so – 16 Q So does the Muscat paper identify an exposure 17 level as necessary to cause harm? 18 A No, they do not. 19 Q Muscat does indicate on, Page 1477, that there 20 were relatively few events in this study and the 21 positive findings could have been due to bias? 22 A Sure. 23 Q What is bias in this context? 24 A Something that is causing the results that is 25 not a true cause. Bias just means that there is</p> <p style="text-align: right;">834</p>
<p>1 significantly with each PCB congener. 2 It is interesting. Not all of them were that 3 way, but starting with – with the 74, lowest tertile 4 was 1; middle tertile was 1.3; and highest tertile 1.7. 5 And then, anyway, they go all the way down. 6 Some of them are statistically significant. Some 7 aren't. The total PCBs is most significant at the 8 highest tertile. 2.9 is the relative risk with the 9 statistical significance. 10 Q This paper actually does contrast with some of 11 the other papers we looked at, even today, which show 12 that some of these same congeners do not increase the 13 risk of breast cancer; correct? 14 A Yeah, I think it would be – they need bigger 15 numbers, probably, to do that, but more importantly – 16 Q Explain that. Who would need bigger numbers to 17 do what? 18 A Well, how many patients did they have? 30 19 patients in the recurrence category. 20 Q You are talking about Muscat? 21 A Muscat. If you had maybe 300, you might be 22 able to start seeing differences in the individual 23 congeners; but they do have mean concentrations in the 24 blood of the various congeners and consistently – 25 pretty consistently, they are higher in the recurrence</p> <p style="text-align: right;">833</p>	<p>1 something that is screwing it up. 2 Epidemiology people always say those sorts of 3 things. It is just terms of epidemiology. 4 It could have been through chance. It could 5 have been bias. We don't know. We tried to remove all 6 the bias; but there is always a risk. Something that we 7 didn't control for. 8 Q Isn't that what epidemiologists spend most of 9 their time doing? Try to eliminate possibility of their 10 chances; influencing their – 11 A Yes, they spend a lot of time. 12 Q That is the whole point. If the result is 13 dictated by chance, then you have wasted your time doing 14 your – 15 A Exactly. You are going to get a negative 16 study. That is why they tighten, over the years, the 17 criteria to say significant. 18 It used to be, when I started out in medicine, 19 P value of .1 was considered significant. Now, it is 20 .05. So you have to have a really good study, really a 21 strong effect to get statistical significance. 22 Q Your next study on your list of breast cancer 23 references is the Negri, N-E-G-R-I, study; is that 24 right? 25 A Yes.</p> <p style="text-align: right;">835</p>

<p>1 Q Handing you what I have marked as Exhibit 147. 2 (Defendants' Exhibits 147 was marked for 3 identification by the court reporter.) 4 BY MR. HOPP: 5 Q This is the Negri study; right? 6 A Um-hmm. 7 Q This is a review article; right? 8 A It is. 9 Q And it looks at exposure to PCB and breast 10 cancer? 11 A Yes. 12 Q And what does Negri and/or her coauthor 13 conclude? 14 A Well, I think the important point is that you 15 need to take into account genetic susceptibility in 16 order to explain what is going on; and that in the 17 general population, without the genetic risk factor, 18 there probably isn't an increased risk. 19 Q So, in fact, at the concluding part of the 20 study, right above the acknowledgments, Negri and 21 coauthors say, "In conclusion, the 22 epidemiological evidence does 23 not support the hypothesis of 24 a direct relation between 25 environmental exposure to PCB</p> <p style="text-align: right;">836</p>	<p>1 of the related compound. 2 Q So it is generally informative, but not 3 directly related? 4 A Correct. 5 Q The next paper in order on your breast cancer 6 reference list is Petralia; is that right? 7 A Yes. 8 Q It is Petralia, 1999? 9 A Yes. 10 Q Petralia has written several articles on this 11 subject; right? 12 A I have Petralia -- another one of the Petralia 13 papers on the new -- that I gave you. Two more, '95 and 14 '98. 15 Q So you have got some older papers? 16 A '98 and '99. So I have got the '99 paper, but 17 I have got an earlier '98 paper that I have added. 18 Q Let me show you Exhibit 148. 19 (Defendants' Exhibits 148 was marked for 20 identification by the court reporter.) 21 BY MR. HOPP: 22 Q This is 1999 Petralia paper? 23 A Yes. 24 Q And Petralia is looking at the premenopausal -- 25 I'm sorry, risk of premenopausal breast cancer in</p> <p style="text-align: right;">838</p>
<p>1 adulthood in the general population 2 and the risk of breast cancer"; right? 3 A That is what he said in the abstract, which I 4 just read to you. 5 Q And then he goes and talks about a 6 specific genetic variation like -- 7 A Right. He is really just repeating what we 8 said earlier about the CYP1A1 and the exon 7. He does 9 not mention exon 7, but in Table 5, he mentioned that. 10 Q But for the general public, Negri is, 11 essentially, a negative paper; right? 12 A Yes, that's the point. But when you take into 13 account the -- see, there is a couple of papers that we 14 have not gone through that are reviewed here that make 15 the same point. 16 Interaction between PCB and the CYP1A1 17 polymorphism, I think what the science has evolved to 18 the point that it takes -- you can have the CYP1A1 gene 19 and not get breast cancer; but if you have it and are 20 exposed to PCBs, then your risk of breast cancer 21 increases significantly. 22 Q And how is this study relevant to Sherrie 23 Barnes? 24 A Well, it is like all of the others. I have 25 referred to in the PCB literature. It shows the effect</p> <p style="text-align: right;">837</p>	<p>1 association with occupational exposure to polycyclic 2 aromatic hydrocarbons and benzene; is that right? 3 A Yes. 4 Q So this is an occupational study? 5 A Yes. 6 Q And does it look at women particularly in these 7 occupations? 8 A It has to be. 9 Q Premenopausal -- 10 A The rate in men, as we know, is quite low. So 11 it is women. And the exposures were variable. 12 They took occupational history of the exposure 13 assessment for PAHs and benzene was developed to 14 determine which occupations had exposure. And then they 15 developed a matrix for that, which included the PAHs and 16 the benzene and others things, as well. 17 Q And which exposure levels did they find to be 18 significant to increase the risk of premenopausal in 19 breast cancer in their occupation when exposed to both? 20 A PAH and benzene, highest risk was in PAH and 21 benzene together. They found statistical significance 22 in all of them and the biggest abnormalities were in the 23 ER positive cases. 24 Q What is that? 25 A Estrogen receptor positive, which we looked at</p> <p style="text-align: right;">839</p>

1 yesterday in our case.
 2 Q Oh, ER-positive breast tumors, that is a
 3 particular type of tumor?
 4 A Yes, this is the first time we have seen that.
 5 Q Okay. Seen what? Seen a study?
 6 A Seen a study where they looked at the ER
 7 positive and ER negative.
 8 Q Forgive me for covering this again, but we
 9 don't know whether Ms. Barnes had a ER positive or ER
 10 negative breast; right?
 11 A Yes, I don't think we do.
 12 Q Again, forgive me for asking you to say this
 13 again, but what was the dose level that the authors of
 14 the Petralia paper found to be significant for inducing
 15 breast cancer? Did you say it was every dose?
 16 A Well, they have got some duration data here
 17 which would be a surrogate for dose.
 18 Q Oh, I see. They use job exposure matrixes and
 19 lifetime occupational history; is that right?
 20 A Yes. And they had low exposure and medium to
 21 high and then cumulative low, medium to high and, in
 22 general, I think they only found a few cases that were a
 23 statistically significant; but the numbers in each cell
 24 are so small that it is not likely to find statistical
 25 significance.

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1 So there were some elevated odd ratios. In
 2 fact, lots of them were elevated; but it didn't reach
 3 statistical significance, except in a few cases.
 4 And, again, if you look at the ends, that is
 5 the problem. There aren't enough in each of the cells
 6 to reach statistical significance.
 7 Q So it is too small a study, really, to
 8 effectively evaluate statistically significant
 9 association?
 10 A With dose. It is a large enough study to say
 11 in general. I mean, you have got quite a few people in
 12 the exposed categories.
 13 Q So overall, looking at overall exposure, they
 14 find an increase risk?
 15 A That's right.
 16 Q But they cannot break that down by exposure
 17 classification?
 18 A Right. Of the patients that they looked at,
 19 they had 25 of PAH alone; 35 of benzene alone; 6
 20 exclusively PAH; 19 PAH and benzene; and 16 exclusively
 21 with benzene. Those are --
 22 Q Small numbers?
 23 A Relatively small numbers, but big enough to get
 24 statistical significance on many of these; but then when
 25 the numbers drop down to 16, 8, 13, and 11, 10 and 9 --

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1 10 and 8, then they don't get statistical significance
 2 even though they have elevated odds ratios.
 3 Q Looking at Page 220, this is the first full
 4 paragraph. It says, "When our results
 5 are interpreted, several issues
 6 need to be considered. The
 7 response rates for both the cases and
 8 referents" -- that is R-E-F-E-R-E-N-T-S
 9 -- "were low."
 10 Now, that is a problem for epidemiology?
 11 A Where are you reading from?
 12 Q Page 220, first full paragraph, starting with
 13 the words, "When our results are interpreted."
 14 A Oh, I see, on the right-hand side.
 15 Q Low response rate is a problem for an epi
 16 study; right?
 17 A Yes, the response rates were low. That is a
 18 problem.
 19 Q And would you characterize it as a case control
 20 study or a cohort study?
 21 A Case control.
 22 Q How does this paper, the Petralia paper, relate
 23 to Sherrie Barnes?
 24 A She was exposed to both benzene and to PAHs,
 25 and so it would be a direct relationship. Although it

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1 was not occupational exposure, she had environmental
 2 exposure. I would submit that she probably had higher
 3 exposures to PAHs and benzene than the people in this
 4 study.
 5 Q Does the Petralia study, then, identify an
 6 exposure level that is necessary to cause harm?
 7 A No, it doesn't have any quantitative data.
 8 Q All right. The next study in order on your
 9 list of breast cancer references is Pliskova; is that
 10 right?
 11 A Yes.
 12 Q P-L-I-S-K-O-V-A. I am handing you what I have
 13 marked as deposition Exhibit 149. This is the Pliskova
 14 paper; correct?
 15 (Defendants' Exhibits 149 was marked for
 16 identification by the court reporter.)
 17 THE WITNESS: Yes.
 18 BY MR. HOPP:
 19 Q I have actually handed you two things. One is
 20 the abstract and one is the article.
 21 A Oh, yeah.
 22 Q Let me have the abstract back, so we don't
 23 confuse ourselves.
 24 A Okay.
 25 Q So Deposition Exhibit 149 is the Pliskova

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1 article; correct?
2 A Yes.
3 Q And Pliskova article states what, specifically?
4 A Benzopyrene and – what is the second one?
5 Benzanthrane.
6 Q Is this an in vitro study?
7 A This is an in vitro study.
8 Q So, again, they are studying cells in a petre
9 dish?
10 A Yes.
11 Q What does Pliskova conclude, if anything?
12 A Well, it is a mechanism paper. They talk about
13 how it induces – benzopyrene induces P53 tumor
14 suppressor expression and abolish both S-phase arrest
15 and apoptosis induced by the PAHs.
16 Potentiated deprecatve effect of BaP. Thus,
17 specific genotoxic and non-genotoxic event for
18 interacting on the effects of BaP cell proliferation.
19 Q How about in layman's term, what are we looking
20 at?
21 A I think the reason that I thought this was
22 important is because of this notation about
23 non-genotoxic mechanisms which hadn't been talked about
24 too much on any other paper.
25 Q Well –

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1 A And they also talk about the BAP and TCDD have
2 some similar toxicities.
3 On Page 254, in the right-hand column, in the
4 first full paragraph, about halfway down it says,
5 "Using a combination of DNA
6 staining and detection of BrdU
7 incorporation, we found that
8 like TCDD, BAP and BAA also
9 partially inhibited induction of
10 S-phase entry by E2. However,
11 unlike TCDD, both BaP and
12 BaA also stimulated G1-S-phase
13 transition, when applied to
14 serum-starved cells, albeit to a
15 lesser extent than E2 itself.
16 Interestingly Diben[a,h]anthracene,
17 a strong AhR ligand, which has been
18 shown to be antiestrogenic in MCF-7
19 cells, had the same effect as TCDD
20 both on the E2-treated and untreated
21 cells. These results seem to support
22 the hypothesis that unlike other PAH's,
23 BaP and BaA, or their metabolites that
24 are less efficient inducers of
25 AhR-mediated activity, can activate ER

845

1 and stimulate cell proliferation."
2 Point being, that they have similar toxicity of
3 TCDD and that the effects of the two together are going
4 to be at least additive.
5 Q Similar effects, when they contact the Ah
6 receptor; right; that is what they are saying?
7 A No, this is talking about other effects. That
8 is the point I was trying to make.
9 Non-Ah receptor stimulated toxicity, because
10 these other types of toxicity to the cells are not
11 related to the age receptor. And they are pointing that
12 out. That's all.
13 Q Let's look at the first page of the article.
14 This is on the right-hand column, about an inch or so
15 down, she says, "Today, PAHs are
16 regarded mostly as antiestrogens
17 principally due to their ability to
18 activate aryl," A-R-Y-L, "hydrocarbon
19 receptor," that is AhR receptor, "which
20 may lead to supression of estrogen
21 response element controlled gene
22 expression."
23 So they are talking about the PAHs being
24 protective in some measure; is that right?
25 A No. Stimulating Ah receptor creates adverse

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1 effect. It influences the ability to the cell to
2 regulate its growth properly.
3 Q Does an antiestrogen cancel out an estrogenic
4 compound?
5 A Yes, it would.
6 Q What is the relevance of the Pliskova paper to
7 Sherrie Barnes?
8 A Well, I have been trying to say that, to me, it
9 addresses the issue of the dioxin, plus the PAHs being
10 more harmful.
11 Q Okay. Does it identify a particular dose or
12 exposure level in which harm would occur?
13 A No. It is an in vitro study. It wouldn't have
14 any quantitative value.
15 Q So it is hypothesis generating in that regard?
16 A No, it demonstrates in an in vitro system, a
17 mechanism. Those give us insights into why we would
18 have this young woman developing such a malignant cancer
19 at such a young age following in vitro, in utero, and
20 early childhood exposure to these chemicals.
21 Q Well, it looked at particular PAHs; is that
22 right?
23 A Yes, they looked at two particular PAHs.
24 Q They did not look at creosote as a mixture?
25 A Right. Correct.

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1 Q Which -- which congeners of dioxin did the
2 Pliskova paper study?
3 A TCDD.
4 Q So you had one congener of dioxin and two
5 different congeners of PAH?
6 A I don't think they actually did TCDD. They
7 just referred TCDD studies. They, themselves, just did
8 PAH studies.
9 Q Okay. So the Pliskova paper does not actually
10 study a synergistic effect between PAHs and TCDD?
11 A Correct.
12 Q It just shows that certain -- certain PAHs at
13 certain levels can have a dioxin-like effect?
14 A There is some missing page here.
15 Q Sorry.
16 A 247 is missing.
17 Q I will have to supply that.
18 A 249 is missing. 251 is missing. 252 is
19 missing.
20 Q You just got the even pages?
21 A So I am looking for things like what they used,
22 but the pages are missing.
23 Q But from the abstract -- and I apologize for
24 that, Doctor. We will supply a full copy when we come
25 back to this in our next session.

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1 From the abstract, it looks like they were not
2 studying the synergistic effect; is that right?
3 A No, they were not studying the synergistic
4 effect. I am simply saying that that is one the reasons
5 why it is relevant.
6 Q It suggests --
7 A It suggested that the two together are going to
8 have a more likelihood of developing the cancer.
9 Q Can we do one more paper and then call it
10 quits?
11 A We will do Revich.
12 MR. HOPP: Keith, you all right? Can you hang
13 in there?
14 MR. PRUDHOMME: Sure.
15 BY MR. HOPP:
16 Q Let's do Revich. The next document on your
17 list, Doctor, 20, is the Revich paper; right?
18 A Yea.
19 Q Handing you what we marked as Exhibit 150.
20 (Defendants' Exhibits 150 was marked for
21 identification by the court reporter.)
22 BY MR. HOPP:
23 Q This is the Revich paper; right?
24 A Yes, sir.
25 Q And Revich is looking at dioxin exposure in

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1 Chapaevsk, C-H-A-P-A-E-V-S-K, Russia; is that right?
2 A Yes.
3 Q What happened in Chapaevsk, Russia to make
4 Revich want to study dioxin exposure?
5 A There was a pesticide plant there that made
6 chlorinated pesticides.
7 Q In particular, TCDD; right?
8 A No. Nobody makes TCDD, but they were making
9 lindane.
10 Q Lindane. Okay.
11 A And they generated a huge pollution with TCDD,
12 TEQs. There was a -- they had levels that are a little
13 bit higher than the levels that we have outside the
14 Koppers plant in Grenada, but not too much higher.
15 There is certainly a good overlap there.
16 Q Okay. All right. Did Revich find an increase
17 incidence of breast cancer in this exposed population?
18 A Yes, I think that is the point. The
19 Chapaevsk -- how did you pronounce it?
20 Q Chapaevsk.
21 A Chapaevsk women had a higher risk overall due
22 to breast cancer. 2.1, at 1.6 to 2.7 and then some
23 other cancers, as well.
24 Increase female breast cancer in all age groups
25 compared to Russia and the Sumara region in 1998. There

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1 is a table in here. I think a graph -- a figure that --
2 Figure 1 and Figure 2.
3 Figure 2 is the female breast cancer rate. 958
4 is the page. And it shows consistently at all ages
5 higher breast cancer rate for women in that region.
6 Q Compared to the rest of Russia and to this
7 other area, the Sumara area?
8 A Yes, which is probably the general area that
9 this thing is in. And they also have some data on
10 concentrations of the PCDD and PCDF in the blood, milk;
11 soil; air; and they also have some data on how far away
12 they were from the plant for concentration of blood.
13 The -- I think this is TEQ -- yes. Picogram
14 TEQ on Table 13, they had six people that they studied
15 which was within one to three kilometers of the plant.
16 Their values were 75, picogram TEQ, opposed to
17 those that were five to eight kilometers away where it
18 was four people. And their value was 24. And then they
19 did some other control values.
20 Q So is this like a cohort study or a
21 cross-sectional?
22 A This is cross-sectional, environmental,
23 biomarker and -- yeah, cross-sectional study. I don't
24 think they had any controls. They used, as I said
25 already, published rates.

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1 Q So did Revich, in looking at breast cancer,
2 attempt to control other known risk factors?
3 A Let me see what he did in terms of that issue.
4 I think he assumed that -- I don't see that he
5 did any analysis, for example, age of menarche,
6 menopause, and all of that other stuff.
7 Q Right. Now, he did identify exposure levels or
8 at least --
9 A Yes, he had exposure levels.
10 Q And did he identify the level at which the
11 exposure is likely to cause harm?
12 A Well, I don't think we can say that because he
13 doesn't have a no effect level.
14 In other words, he has a level of blood TEQs in
15 six people that lives within one to three kilometers of
16 the center. So we can say that if you are between the
17 background level and that level, somewhere in there
18 would be the level at which you start seeing any
19 excesses.
20 Q He does not give us a bright line for excess
21 levels of cancer?
22 A Well, what they say in regulatory circles is
23 that he gave a single value that was the only and,
24 therefore, the lowest observed adverse effect level of
25 75 in the blood.

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1 A Yeah, that is the workers. I am talking about
2 people who were living next to the site.
3 Q One to three kilometers, it was 75.2?
4 A That's right.
5 Q And for his analysis of breast cancer, does he
6 combine the workers and women who lived near the plant
7 or does he examine just the women who lived one to three
8 kilometers from the plant?
9 A I think he may have combined them, but I don't
10 know, looking at this. Yes, that was four workers who
11 worked in the plant. And that he refers to an earlier
12 paper that he published that report.
13 Q Six women who lived from one to three
14 kilometers?
15 A And there was six women who lived between one
16 to three kilometers. That is where the 75 came from.
17 That's also from an earlier paper.
18 Q So workers from one to three kilometers
19 combined, that is a total of ten women; right?
20 A Yes. Well, it does not say that they are all
21 women; do they?
22 Q Yeah, look at Table 4. Female blood?
23 A One to three kilometers. Four -- okay. The 75
24 is the one -- is the six there?
25 Q Maybe it is a bit obscure, I mean, the title of

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1 Q 75 picograms per gram?
2 A Yes, picograms per gram.
3 Q And that is total TEQ?
4 A Total TEQ.
5 Q Which is much higher than the level that was
6 measured in the cohort in Grenada; correct?
7 A Well, it is a -- it is higher than the average.
8 Q Total TEQ in Grenada was 34; right?
9 A Not the highest values, no. I think we had
10 some others that were high.
11 Let's see if I can find where I did look at
12 this. We have one -- I think one of these values. The
13 TEQ was 92 on one of our folks and another one was 93.
14 One had 50. One at 89. So we had some that were
15 clearly up in that range.
16 Q Okay. But --
17 A Yeah, the mean value is whatever we said it
18 was.
19 Q But what is the mean value in the Revich paper?
20 A 75. They didn't give the breakdown. Yeah, the
21 mean value is higher. I agree with that. Now, there is
22 also some soil values here.
23 Q Well, let's start with the workers -- female
24 workers' blood. The workers had a total TEQ of 412; is
25 that right?

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1 the table says Female Workers Blood and then the column,
2 one to three kilometers, that is not workers. So it is
3 a bit ambiguous; isn't it, with respect --
4 A Yeah, it is. That is an interesting question.
5 Are they all women or is this men and women?
6 Let's see, we are talking about dioxin and public
7 health. The guy is not a really skilled writer.
8 Q Well, he is Russian.
9 A Well, it is not his native language. It's hard
10 for them to sometimes get it straight. Even I have had
11 Russian papers that I read and had them translated, and
12 they were really awful.
13 But here is an example of some complexity that
14 is hard -- blood samples were taken from 14 people.
15 90 percent of women lived in Chapaevsk versus for more
16 than three years. So maybe it is all women.
17 Q Total on Table 4 is 14. You got four workers,
18 six --
19 A Yeah. 90 percent of the women -- it must be
20 all women.
21 Q Okay.
22 A But it does not say that anywhere.
23 Q All right. In any event -- at any rate, Revich
24 identifies the effect level being 75 picograms per --
25 A Yeah. Yeah. We do have something to look at.

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<p>1 MR. HOPP: Okay. All right. Shall we knock 2 off for the day? It is 5:00 o'clock.. 3 THE WITNESS: You won't get an argument out of 4 me. 5 /// 6 /// 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25</p> <p style="text-align: right;">856</p>	<p>1 I, the undersigned, a Certified Shorthand 2 Reporter of the State of California, do hereby certify: 3 That the foregoing proceedings were taken 4 before me at the time and place herein set forth; that 5 any witnesses in the foregoing proceedings, prior to 6 testifying, were placed under oath; that a verbatim 7 record of the proceedings was made by me using machine 8 shorthand which was thereafter transcribed under my 9 direction; further, that the foregoing is an accurate 10 transcription thereof. 11 I further certify that I am neither 12 financially interested in the action nor a relative or 13 employee of any attorney of any of the parties. 14 IN WITNESS WHEREOF, I have this date 15 subscribed my name. 16 17 18 19 20 21 22 23 24 25</p> <p style="text-align: right;">Dated: _____</p> <p style="text-align: right;">_____ Diana Janniare CSR No. 10034</p> <p style="text-align: right;">858</p>
<p>1 2 3 4 5 6 7 I, JAMES DAHLGREN, M.D., do hereby declare 8 under penalty of perjury that I have read the foregoing 9 transcript; that I have made any corrections as appear 10 noted, in ink, initialed by me, or attached hereto; that 11 my testimony as contained herein, as corrected, is true 12 and correct. 13 EXECUTED this _____ day of _____ 14 _____ 15 20__, at _____ 16 _____ (City) _____ (State) 17 18 19 20 JAMES DAHLGREN, M.D. 21 22 23 24 25</p> <p style="text-align: right;">857</p>	<p>1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25</p>

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